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Desensitization in Bronchial Asthma.—Skin tests according to Coca's technique were made on 300 asthmatic patients. Thirty-two different extracts were used. Strongly positive reactions were observed in 8 per cent of the patients out of a total of 4,023 intracutaneous tests. The most frequent reactions were obtained with extracts from dust, feathers from bedding and various food stuffs. In spite of frequent positive tests with food proteins the impression was gained that these proteins play an unimportant aetiological rôle. The most important allergens are apparently dust, feathers and pollen. It is concluded that a complete testing should be done only on asthmatics in whom the usual treatment does not bring any relief, and that in such patients testing with a very large number of different extracts is necessary to determine correctly the causative agent. Out of the 300 asthmatics, 116 were treated a sufficiently long time to permit conclusions as to the efficiency of the treatment. The various methods of treatment were as follows: In every case in which a specific allergen could be established as a causative agent it was attempted to remove this particular allergen from the patient. When this was impossible specific desensitization was attempted. In patients in whom no specific allergen could be found as the cause of the disease, nonspecific desensitization with tuberculin or with peptone was tried, or sulphur therapy, or psychotherapy. All patients were observed for a minimum of two years. Complete freedom from attacks was obtained in 24 per cent of patients treated with peptone, in 26 per cent treated with tuberculin, in 30 per cent treated with dust extracts, in 26 per cent treated with dust extracts and peptone, in 44 per cent treated with dust extracts and tuberculin, in 36 per cent treated with psychotherapy and in 100 per cent of those patients in which the causative allergen could be banished from the surroundings of the patients. It is concluded that the value of skin tests for the recognition of specific allergens in the causation of asthma has been greatly overvalued. It is usually sufficient to use for these tests only extracts from feathers, dust and in certain cases from pollen. Since nonspecific desensitization is frequently successful it is not necessary in every case to search for the specific allergen. This should be done, however, in all cases which resist the usual desensitizing therapy. The best treatment is to prevent contact between the patient and his specific allergen. The nonspecific desensitization with tuberculin or peptone yields about 60 per cent of markedly favorable results.—*Zur Desensibilisierungstherapie des Asthma Bronchiale, H. Petow und E. Wittkower, Klin. Wchnschr., September 13, 1930, ix, 1712.*—(*M. P.*)

Empyema in Children.—In analyzing the results of a method of treatment certain factors should be considered in every case, as follows: *1:* The condition of the abscess and surrounding tissues. *2:* The time-relation of the formation of the empyema to the original pneumonia, which is the most important single factor in prognosis. *3:* The duration of the empyema before treatment is begun. *4:* The results at least a year after cessation of treatment, confirmed by X-ray examination. *5:* The age of the patient, as children

are more liable to have the synpneumonic type of empyema which forms while pneumonia is still causing critical illness. *6:* The type of the infecting organisms, since synpneumonic empyema is produced by the streptococcus more often than by the pneumococcus. *7:* Complications, such as pericarditis and metastatic abscesses, the incidence of which increases with the weakness and youth of the patient. *8:* Intercurrent diseases, such as measles and ear infections, which increase the mortality. Empyema is never cured until its cavity is obliterated by symphysis of its granulating pleural walls. Opening the pleural space of a normal chest embarrasses respiration through the inrush of air on inspiration rather than because of the collapse of the lung, as the degree of dyspnoea and the size of the opening are in direct proportion. Opening an empyema with relief of the pressure of the pus allows the lung to reëxpand to a degree depending upon the condition of the lung and pleura, but unless the empyema walls are stiffened the effect during inspiration may be almost as serious as in the normal chest. Too rapid removal of the pressure which the pus exerts on the sensitive thoracic viscera may cause serious shock. In the synpneumonic type the walls have not had time to stiffen, and the pus should be aspirated slowly at intervals frequent enough to prevent pressure effects on the thoracic viscera, thus delaying continuous drainage until a favorable time. Blood transfusions are valuable. *Débridement* of an empyema is dangerous and unnecessary, especially since a true loculated empyema is rare. The open and closed methods of drainage are discussed and evaluated as too extreme. The method recommended is as follows: The empyema cavity is entered, preferably through the 6th or 7th interspace in the posterior axillary line. It is a mistake to aim for the reflection of the pleura on to the diaphragm. Two dePezzer self-retaining catheters are inserted. The wound is dressed with gauze soaked in liquid paraffine and the pus is not evacuated until the child has rested for an hour or so in bed on a canvas sheet laced to a frame. The chest sags into a hole in the sheet and the lower catheter drains into a bottle of antiseptic solution under the bed. Irrigation is done with warm Dakin's solution every three or four hours. Blowing exercises are begun when the temperature is normal. Walking is usually begun after two weeks. The catheters are removed one at a time when the cavity holds less than 10 cc., irrigations being continued.—*Treatment of Empyema in Children, D. Browne, Lancet, October 4, 1930, ccxix, 733.*—(*R. M. F.*)

Lung Abscess.—The author presents a study based on 35 cases of nontuberculous abscesses of all types. There is considerable experimental and clinical evidence to show that, of chronic infections in the upper respiratory tract, the retention in the lungs of certain anaerobes ordinarily found in the oral cavity, and preëxisting bronchopulmonary pathological change, each plays a considerable rôle in the production and chronicity of abscesses. Likewise, anything that tends to abolish the cough reflex during anaesthesia or to increase cough after anaesthesia may be instrumental in production and chronicity. Statistics show that the lower lobes are more frequently involved than the upper, and the right lung more frequently than the left. Generally speaking, the symptoms are out of proportion to the changes observed on physical examination. The roentgen ray is perhaps the most important single factor in the recognition of an abscess. In its early stages the abscess is frequently seen as an acute irregular consolidation, and may resemble a pneumonic tuberculous consolidation, interlobar empyema or a new growth. The prognosis will depend on the cause, type, location and duration of the abscess. Apical and hilar abscesses have a better outlook than central or peripheral ones, while acute have a better prognosis than chronic abscesses. The author carried out the following line of treatment: Patients were kept at complete bed-rest during the acute stage, and postural drainage was instituted several times a day, depending on the tolerance of the patient to this form of treatment. Patients were given soft but nourishing foods and medication to control any annoying symptoms. Expectoration was encouraged. If no improvement ensued within a reasonable length of time, and if the abscess was not peripherally placed, pneumothorax was attempted. If not successful, more radical surgery was resorted to when possible. Bronchoscopy, transfusions and arsphenamine were not used to any extent. After the subsidence of acute symptoms, patients were discharged to continue their treatment at home, being urged to remain in bed most of the day as long as physical and roentgen-observations indicated that a diseased condition was still present. They were at first treated like acute, and later as quiescent cases of tuberculosis. Of the entire 35 cases, 19 were either improved or cured, while 16 died. The following conclusions are drawn: All lung abscesses are primarily medical. Operation during the acute phase of abscess carried with it a mortality of from 65 to 70 per cent. Acute single lung abscesses and some chronic abscesses are usually amenable to medical treatment alone.

Radical treatment is definitely indicated only in cases in which the patient does not get well after a reasonable period of conservative management, in peripheral abscesses that do not drain well, in long-standing chronic cases, or, occasionally, in multiple abscesses limited to one lobe. Bilateral multiple abscesses present a virulent condition, running a rather brief and stormy course and leading to a fatal termination. Early diagnosis and early medical treatment are the secrets of success in the cure of solitary lung abscesses.—*Lung Abscesses: A Clinical Study, with Especial Emphasis on Conservative Treatment, H. I. Spector, J. Am. M. Ass., September 13, 1930, xcv, 789.*—(*G. L. L.*)

Suppurative Diseases of Lungs.—The primary factor from the bronchoscopist's point of view in all suppurative diseases of the lung is bronchial obstruction. One type is complete blocking of the bronchus. The two others are different kinds of valvular stenosis. The three types are likened to the action of valves in hydraulics: the by-pass valve, the check valve, and the stop valve. In the first, the lumen is diminished, but air can still pass in both directions. In the second, air can flow only in one direction because the walls of the bronchus are compressed by the obstruction so that the air cannot escape, though when a very deep breath is taken, air can be drawn past the obstruction, the lumen closing again at the beginning of inspiration. The third or stop valve represents complete obstruction. The check-valve action of the bronchus is due to the clogging of the cilia by suppurative material, rendering the cough reflex less efficient. Bronchoscopic aspiration can be used to restore the defensive power of the lung, especially the ciliary action, and can prevent stagnation of pus which is the chief factor in the production of a chronic condition, arising out of an acute suppurative process.—*Suppurative Diseases of the Lung, C. Jackson, Brit. M. J., October 25, 1930, no. 3642, 702.*—(*M. E. P.*)

Treatment of Suppurative Pneumonitis.—The lesions of the earliest stage of suppurative pneumonitis are usually a purulent infiltration of the alveoli and interstitial tissue of the lung with an associated infection of the bronchioles and smaller bronchi. The causes of the lesions are many and have an obvious bearing on prophylaxis and treatment. Chief among the causes are the following: (1) aspiration of infected material, which most commonly comes from infected mouths or sinuses; (2) aspiration of any type of foreign body which may infect the lung; (3) prolonged bronchial obstruction by neoplasms, aneurysms, strictures, foreign bodies and mucous plugs, which may cause suppurative pneumonitis through stasis of trapped secretions; (4) incomplete resolution of pneumonia; (5) bacteriaemia; (6) pulmonary infection from extension of infection through the lymphatics from neighboring foci; and (7) wounds of the lung, especially when a foreign body remains. In the early stages of the disease, medical treatment is best and often cures. If it fails after a reasonable trial, surgery frequently offers an excellent chance for cure, provided operation is not unduly delayed. The most useful medical and surgical methods of treatment are sanatorium regimen, frequent postural drainage, elimination of mouth and sinus infection, neoarsphenamine given intravenously; bronchoscopic removal of foreign bodies and secretions, artificial pneumothorax, temporary or permanent interruption of one phrenic nerve, extrapleural pneumolysis, extrapleural thoracoplasty, drainage of an abscess, and lobectomy. Selection of the best therapeutic procedure for each of the types of suppurative pneumonitis must be highly individualized. The simplest appropriate measure should be chosen first, and as soon as it is seen to be inadequate the next more radical one used. In spite of faithful medical treatment, suppurative infiltration may become an abscess or multiple abscesses, or may become gangrenous. Even now, if little time has elapsed, continuation of the same treatment will bring about a cure in perhaps 30 per cent of cases. Bronchoscopic aspirations may be given a fair but not too prolonged trial. Instillations of iodized oil have not sufficiently proved their value. The authors have had no experience with intrabronchial neoarsphenamine. If the abscess still persists, a temporary phrenic-nerve interruption should be performed. This operation has served better than any other measure in the control of all degrees of haemoptysis in abscess, as well as in bronchiectasis. A few cases of abscess that persist may be treated by artificial pneumothorax, when the results are occasionally astonishingly good. Pneumothorax should not be used for those abscesses that dwell in the peripheral portion of the lung, because here adhesions may tear the infected lung and be responsible for a highly dangerous form of empyema. The lung and its contained abscess cavity may be relaxed or compressed locally by extrapleural pneumolysis, which may also prove valuable in severe haemoptysis. Eight or ten weeks after the onset of the disease, if the patient is not clearly showing signs of improvement, surgical drainage of the abscess must not be delayed. Several aspects of operation are con-

sidered important: (1) local anaesthesia is superior to inhalation narcosis; (2) the axillary is preferable to the anterior approach in most abscesses of the upper lobe; while the posterior or posteriolateral is the obvious approach to the lower lobe; (3) removal of ribs wide of the limits of the abscess is preferable to restricted resections; (4) one of the greatest dangers of the operation is infection of the pleurae when the layers are not completely and firmly adherent in the field of operation; (5) the use of a rubber tube to drain an abscess may cause a dangerous haemorrhage. It is best to cauterize a wide opening to the abscess until the cavity has filled in from its very bottom. Extrapleural paravertebral thoracoplasty is not indicated in any but the chronic stage of abscess; here its best results are obtained in abscesses with thin compressible walls or in small single or multiple abscesses. Certain very extensive multiple abscesses cannot be well drained in the ordinary sense. For them some type of lobectomy should be considered. Some cases of noncavernous suppurative pneumonitis progress to bronchiectasis. Therapeutic procedure for bronchiectasis varies greatly according to the following conditions: (1) whether the lesions are limited to one or more lobes; (2) the amount and foulness of the sputum; (3) the general condition and age of the patient; and (4) the frequency of fresh attacks of bronchopneumonia. The *bronchiectasis sicca* of Bezançon is commonly associated with a fibroid lung and displacement of the mediastinal organs. The authors believe that phrenicectomy is indicated to help the contraction of the fibrous tissue within the lung. Bilateral bronchiectasis should not be treated radically. Most important of all in palliative treatment is the persistent use of postural drainage every few hours. When the disease is confined to one lower lobe phrenicectomy should be the first measure to be tried. In occasional early cases it alone may cure. Artificial pneumothorax may cure an early but rarely a late case. Extensive extrapleural thoracoplasty has its principal value in (1) the rather rare type of bronchiectasis that is limited to the smaller or peripheral bronchi that are compressible because of their incomplete cartilaginous framework and (2) bronchiectases that are accompanied by great traction-displacement of the mediastinal organs. If, after all above measures have been tried, the patient continues to have abundant foul sputum, and remains in a toxic condition, a lobectomy must be considered. The operative mortality of two-staged lobectomy ranges between 20 and 30 per cent in the most experienced hands. The improvement that follows total removal of the only diseased lobe is truly remarkable. Such patients may be classified as completely cured. For cases in which the pleurae over the diseased lobe are extensively adherent, the authors recommend the staged cautery lobectomy of Evarts Graham. For cases in which the pleurae of the diseased lobe are not extensively adherent, they believe that the exteriorization lobectomy described by Whittemore is the operation of choice.—*Treatment of Non-Tuberculous Suppurative Pneumonitis, Abscess and Bronchiectasis, J. Alexander and W. W. Buckingham, J. Am. M. Ass., November 15, 1930, xcv, 1478.*—(*G. L. L.*)

Amoebic Abscess of Lung.—Among 5 cases of amoebic abscess of the liver there was one case in which an abscess presented in the upper lobe of the right lung. Such a secondary abscess may be caused by blood-stream infection, but in this instance it was apparently due to an extension of the liver abscess through the diaphragm into the pleural space and thence into the lung. There had been loose watery stools prior to the pulmonary illness. The stools contained *Endamoeba histolytica*, as did the pus from the thoracic cavity. Improvement followed the administration of two courses of emetine and an arsenic compound similar to acetarsone. Thoracotomy was performed and a pleural pocket was opened. On its inner side was necrotic lung tissue, which also contained amoebae. Recovery followed two more courses of emetine.—*Amebic Hepatic, Subphrenic and Pulmonary Abscesses, S. W. Harrington, Arch. Surg., December, 1930, xxi, 1146.*—(*R. D.*)

Hydatid Cysts of Lung.—This paper calls attention to the possibility of hydatid disease originating in American-born persons. In South America the disease is increasing in incidence, and in Australia it shows no reduction in frequency. There is reason to fear that there may be a serious increase of the disease in this country, although its distribution in domestic animals is as yet very patchy. Next to the liver the lung is the most common organ to be invaded by *Taenia echinococcus*. Following ingestion the embryo passes through the intestinal wall and reaches the blood-stream. By this means it is conveyed to the lungs. It is also possible that the embryo reaches the lung by the respiratory route. Pulmonary cysts grow rapidly as the surrounding tissue does not resist their expansion. The cyst depends on osmosis for nutrition. The majority of the cysts rupture sooner or later. Erosion into a blood-vessel is a common occurrence, but rupture into a bronchus is more frequent. The contents of the cyst may be

expectorated, but secondary infection is more common. Cysts located in the periphery of the lung may rupture into the pleura producing a hydrothorax. In the symptomatology cough is most important; haemoptysis is common; pain from pleural irritation is almost as frequent; and dyspnoea occurs when the cyst attains a large size. In the unruptured cyst the X-ray shows characteristic oval or rounded shadows, multiple or single, with clear-cut edges, and this picture in a patient presenting the above symptoms is almost diagnostic. Complement fixation and intracutaneous tests may be used for confirmation. Eosinophilia is inconstant. After rupture of an intrapulmonary cyst additional diagnostic aids are available. Following the general symptoms noted above, the patient, during a coughing spell, finds the air-passages flooded with a watery expectoration which has a salty taste. Portions of the cyst-wall or contents of the cyst, notably hooklets from degenerated scolices, may be found in the sputum. If the cyst is incompletely emptied a fluid level shows on the X-ray film. Exploratory needle puncture is severely condemned except for hydrothorax. There is no medical treatment for the condition. Expectant treatment is justifiable in children or if the cysts are small and deeply located. A large proportion of cases rupturing into a bronchus go on to recovery. Peripheral cysts of large size require surgical intervention. It is to be reiterated that aspiration is dangerous. In cases not secondarily infected the procedure is not standardized. The ideal is extirpation of the cyst and closure without drainage, but two-stage procedures may be necessary and, if there is a thick adventitia, suture to the chest-wall and drainage are advised. Injection of formaldehyde before incision is unwise because of danger of the solution entering the bronchi. The ruptured and secondarily infected cysts are dealt with by the surgical measures employed in pulmonary suppuration. The progress in cases without multiple infestation is favorable in about 80 per cent. Of two cases here reported one had always lived in New York State, the other had lived in Italy. Thirty-four other cases occurring in North America are summarized.—*Hydatid Cysts of the Lung, E. W. Phillips, Arch. Surg., December, 1930, xxi,* 1324.—(*R. D.*)

Hydatid Cyst of Lung.—A woman, aged 26, complained of pain in the posterior thorax. Examination revealed a soft, fluctuant mass, not adherent to the skin. Exploratory puncture was done and a watery fluid was withdrawn. Further examination disclosed that it was a hydatid cyst and at operation it was located subcutaneously and communicated with a deep cavity, the size of an orange, within the thorax. The patient recovered.—*Kyste hydatique du poumon à extériorisation thoraco-pariétale, Bressot, meeting Soc. des Chirurgiens de Paris, November 5, 1929, reported in Progrès méd., November 30, 1929, no. 49, 2111.*—(*U. E. Z.*)

Aetiology of Primary Bronchiectasis.—This paper considers only those cases of nontuberculous ulceration and dilatation of the larger bronchi characterized by a chronic course, distressing cough, and large amounts of sputum which may be very foul. Primary bronchiectasis is analogous to aneurysm in many respects. Both arteries and bronchi are hollow muscular tubes, the main strength of which is derived from the dense sheet of elastic tissue in their walls. Arteries cannot dilate until the integrity of the elastic-tissue layer is destroyed by the action of *Spirochaeta pallida.* Similarly, Smith believes, the bronchi present the sacculated, cylindrical or fusiform dilatations characteristic of primary bronchiectasis only after the elastic-tissue fibres have been weakened and ruptured by the action of an anaerobic group of organisms which includes among its members *Treponema microdentium* and *Treponema macrodentium. Summary and Conclusions:* The essential lesion in primary bronchiectasis is a focal necrosis of the elastic tissue in the bronchial wall due to infection with the fusospirochaetal organisms which are found in pyorrhoea alveolaris, Vincent's angina, pulmonary abscess, and pulmonary gangrene. These organisms have been seen in the sputum of 49 of 60 patients proved to have bronchiectasis by the iodized-oil test. Spirochaetes and fusiform bacilli were found in sections of bronchial dilatations after staining by Levaditi's method in 8 of 12 cases which came to necropsy. Fusiform bacilli were demonstrated in pathological sections from 4 of an additional 6 cases after staining by Goodpasture's method. In animal experiments, fusospirochaetal material from varied sources (pyorrhoea alveolaris, fusospirochaetal bronchitis and pulmonary abscess) caused bronchiectasis as well as pulmonary abscess and pulmonary gangrene in rabbits. Control inoculations with pure cultures of *Staphylococcus aureus,* aerobic haemolytic streptococci, anaerobic haemolytic streptococci, green-producing streptococci, anaerobic streptothrix, Friedlander's bacilli and influenza bacilli failed to produce damage to the bronchi. The fusospirochaetal group of organisms is probably the cause of primary bronchiectasis.—*Etiology of Primary Bronchiectasis, D. T. Smith, Arch. Surg., December, 1930, xxi, 1172.*—(*R. D.*)

Mediastinal Pleurisy and Bronchiectasis.—Many cases of bronchiectasis show on roentgenographic examination a triangular basal shadow ascribed by Rist and others to a mediastinal pleurisy. The lesion responsible for such an image is either a splenopneumonia or a deeply seated peribronchiectatic corticopleuritis of the lower lobe. Many of these shadows clear by spontaneous absorption.—*Essai d'interprétation des images radiologiques triangulaires dites de pleurésie médiastine, se superposant aux bronchiectasies, Besançon, Azoulay, Weismann, Netter and Oumansky, meeting Soc. Med. des Hôp., November 29, 1929, reported in Progrès Méd., December 14, 1929, no. 51, 2207.*—(*U. E. Z.*)

Penetrating Wounds of Pleural Cavity.—The commonest wounds of the chest as met with in civil life differ in type from the extensive wounds met with during the World War. Those occurring in civil life are usually the result of pistol bullets or stabbing with a knife. They seldom require surgical exploration of the pleural cavity. Of 162 cases 47 were stab wounds and 115 gunshot wounds. In 27 cases associated injuries of the abdomen, spinal cord, or skull caused death. Only 7 patients died of the 135 who had penetrating wounds involving the chest alone. Three of these died of haemorrhage from the heart. In 6 patients bullets were removed from the lung, but in no instance before two weeks had elapsed after the injury. In dealing with haemothorax, it is of note that blood in the pleural cavity does not clot readily. With an extensive haemothorax the patient is sick, often with a temperature reaching 103°F. Furthermore, the presence of haemothorax is conducive to the establishment of an empyema. It should not be allowed to remain. Methods of removal include simple aspiration; aspiration and immediate replacement with air; and allowing the blood to leak out of the closed wound in the chest-wall by putting the patient in a position where this point is dependent. The latter method was used in 38 cases and gave good results.—*The Treatment for Penetrating Wounds of the Pleural Cavity, D. S. Allen, Arch. Surg., December, 1930, xxi, 1161.*—(*R. D.*)

Atelectasis.—In at least one case after each five to ten operations, some degree of atelectasis occurs. The reason for the occlusion that so often develops is to be found in the acapnial position of the lungs as a consequence of pain, anxiety, operation and anaesthesia. The causal sequence leading to surgical shock and that leading through atelectasis to postoperative pneumonia are closely related. Both originate in acapnia, one through depression of the circulation, the other through depression of respiration. Acapnia is closely related to asphyxia. Either of these deficiencies, in the one case carbon dioxide, in the other oxygen, disturbs the respiratory processes of the tissues, and each involves a considerable degree of the other. A slight degree of acapnia may be induced by overventilation of the lungs. A more intense and dangerous form may result from excessive breathing in badly administered anaesthesia. In the most severe forms of acapnia leading up to surgical shock, inhalation of carbon dioxide effects the restoration of the alkali bicarbonates and carbon dioxide content of the blood. Depression of circulation after operation and anaesthesia is due to the lowered activity of the respiratory and other nerve centres that influence skeletal muscles. The result is an atonic condition of all the muscles of the body and a decrease of muscular pressure on the tissues which permits the blood to stagnate in the venules and decreases the venous return to the heart. This depression of the normal venopressor mechanism is counteracted by inhalation of carbon dioxide. The consequent increase of muscular tonus augments the venous return and restores the volume of the circulation. After every major surgical operation there is not only a decrease in the volume of air breathed but also a prolonged loss of tonus and relaxation in the thoracic muscles and the diaphragm. The vital capacity of the thorax is thus greatly decreased; the lungs are correspondingly deflated, and occlusion of pulmonary airways rapidly develops. Air in occluded lobules, lobe or lung, is then absorbed, and atelectasis is produced. If pathogenic organisms are present they find in the unaerated undrained area conditions favoring their growth, and pneumonia may result. Inhalation of carbon dioxide is thus a specific preventive of the postoperative pulmonary complications that lead to pneumonia.—*Acapnia as a Factor in Postoperative Shock, Atelectasis and Pneumonia, Y. Henderson, J. Am. M. Ass., August 23, 1930, xcv, 572.*—(*G. L. L.*)

Persistent Congenital Atelectasis.—Two cases of congenital atelectasis are reported. They had the X-ray appearance of disseminated infiltration in the medial portion of the pulmonary field, the periphery of which was clear. One died at two months of age, the other at nine months, each in an attack characterized by extreme cyanosis, cough and labored respiration after several previous similar attacks. The diagnosis was clear from the clinical standpoint in one, but

a diagnosis of unresolved bronchopneumonia was made in the other. In these cases differential diagnosis had to be made as between atelectasis and chronic bronchitis, bronchial asthma, bronchopneumonia and tuberculosis. Repeated roentgenograms on these children consistently revealed the same findings. Emphysema of the remaining functioning tissue probably was responsible for the fact that the plates did not reveal much more marked consolidation. Autopsy revealed large, almost solid, noncollapsible lungs, with marked smallness of the alveoli and absence of exudate. About 95 per cent of the alveoli contained large vacuolated cells.—*Persistent Congenital Atelectasis, C. B. Rose, Radiology, May, 1930, xiv, 488.*—(*H. S. W.*)

Acute Postoperative Pulmonary Collapse.—Three days following an appendicectomy, a boy, aged 6 years, complained of a sudden sharp pain in the chest. Breathing was diminished over the entire side, with absolute dulness. Dyspnoea was urgent. The next day there was marked retraction and immobility of the entire hemithorax. Subjectively the patient felt better. Within a few days all signs disappeared and recovery was uneventful.—*Collapsus aigu du poumon post opératoire, Mocquot, meeting Soc. de Chirurgie, November 6, 1929, reported in Progrès Méd., November 23, 1929, no. 48, 2068.*—(*U. E. Z.*)

Diaphragmatic Hernia.—The present status of the roentgenological diagnosis of diaphragmatic hernia may be summed up as follows: *1:* The incidence of the condition is higher than it has been assumed, and hernias at the oesophageal hiatus are relatively common. *2:* Hernia should always be considered in examinations of the thorax and especially when anomalous shadows are seen in the lower thoracic fields. *3:* In examining the stomach, hernia cannot be excluded without the employment of recumbent postures and manual pressure over the abdomen. *4:* It cannot be excluded without examination of the large bowel, for the colon alone may be implicated. *5:* Its distinction from other lesions depends on the demonstration and identification of abdominal viscera above the diaphragm; neither normal nor altered respiratory movements of the diaphragm are reliable as differential criteria. *6:* The clinical significance of small, impermanent hernias at the hiatus which are demonstrable only under more or less artificial conditions has not been fully determined.—*Progress in the Roentgenologic Diagnosis of Diaphragmatic Hernia, A. B. Moore and B. R. Kirklin, J. Am. M. Ass., December 27, 1930, xcv, 1966.*—(*G. L. L.*)

Hydropneumopericardium.—A case is reported of hydropneumopericardium in a man, aged 61, who was admitted to the hospital three weeks after the onset of severe pain over the heart, accompanied by dyspnoea. The heart-sounds were obscured by loud ringing metallic splashes, heard in the upright position only. The above diagnosis was made, but not confirmed by roentgenograms. The patient recovered. The condition was explained by erosion following adhesive tuberculous pleuropericarditis, there being fibroid tuberculosis of both upper lobes. From a study of the literature on the 77 reported cases, it is found that 75 per cent of these conditions are caused by perforation of the pericardial cavity from without. The physical signs established the diagnosis. There is a high-pitched, tympanitic note over the precordium with the patient in a recumbent position, and auscultation in certain positions gives the characteristic metallic splashing sound, often audible at a distance of several feet. Two-thirds of the total cases in which the result is known terminated in death. The prognosis varies with the aetiology, traumatic cases showing the greatest percentage of recoveries.—*Hydropneumopericardium: Report of a Case with a Summary of the Literature, R. T. Shackelford, J. Am. M. Ass., January 17, 1931, xcvi, 187.*—(*G. L. L.*)

Pulmonary Embolism in Infant.—Nine days following a mastoid operation an infant 7 months old became acutely ill and died four hours later. At postmortem examination an old rounded clot was found blocking the branches of the pulmonary artery to both lower lobes and the right middle lobe. In the literature there are only 11 cases of pulmonary embolism in childhood, 8 of which were due to aural infection.—*Pulmonary Embolism in an Infant following Aural Infection, J. A. Jones, Lancet, November 15, 1930, ii, 1070.*—(*B. T. McM.*)

Psittacosis.—The historical aspects, epidemiology, clinical picture, pathological findings and aetiology of psittacosis are reviewed. The first recorded epidemic occurred in Switzerland in 1879 and the disease was then recognized as presenting symptoms and signs of both pneumonia and typhoid fever. Nocard found a short, gram-negative bacillus, resembling organisms of the Salmonella group, in the bone-marrow of dried wings of parrots who had died of this disease. The evidence in favor of occasional human transmission seems to be unassailable. No cases have occurred in which there has been no association with infected birds or patients. It is not an invariably fatal disease and one

attack possibly confers lifelong immunity. The incubation period is about ten days and the duration of the disease is usually two or three weeks. Convalescence is protracted and may be interrupted by relapses or by thrombosis of the femoral vein. High fever, headache, epistaxis, sore throat, chills, generalized pain, profound exhaustion, and paroxysmal cough with mucoid or purulent sputum occur early. Patchy areas of consolidation appear in the lungs. The respirations are not increased markedly and the pulse-rate is relatively slow. Delirium and semi-comatose states may alternate. A "typhoid state," with rose spots on the skin and involvement of the gastrointestinal tract, is common. The mortality rate is about 20 per cent. The pathological findings are those of a generalized septicaemia with an inflammatory condition of the lungs. Intense engorgement, proliferation and desquamation of epithelium, leucocytic infiltration and increasing haemorrhage are first found in the lungs. This is followed by an abundant, serous and fibrinous exudate, with an increasing capillary thrombosis. The consolidation is of the lobar type. Areas of degeneration and haemorrhage are found in the muscles, chiefly the rectus abdominis. The kidneys and liver show parenchymatous degeneration, and a diffluent red septic spleen is a common finding. Congestion and oedema of the brain and spinal cord are not infrequent. Bedson and Western have concluded from experimental work that the disease is due to a filterable virus. Gordon has shown that strains of the virus increased in virulence by passage through mice. Dermotropic and neurotropic affinities displayed by this virus for rabbits, after passage through mice, suggest that it may belong to the same group as the herpes virus.—*Psittacosis, Ministry of Health Report, Brit. M. J., December 13, 1930, no. 3649, 1018.*—(*M. E. P.*)

Pneumonoconiosis.—The clinical picture of pneumonoconiosis is already mentioned by Hippocrates and is well described by Paracelsus in 1534. Ramazini, in 1703, gave a complete description of the disease although he undoubtedly included some cases of pulmonary tuberculosis. In the sixteenth century it is reported that the mortality from pulmonary diseases of miners in the Karpathian Mountains was so great that many women in these regions married as often as seven times, and it is a well known saying that the Sheffield miner never sees his grandchild. In the beginning of the nineteenth century a German cleric, Götzinger, wrote a travelogue about the Saxonian Mountains and mentions that most of the laborers employed in the quarries die between 30 and 40 years of age and that only an occasional one reaches the age of 50. Tool-grinders in industrial plants in the years 1850–1870 had such a high mortality that 82 per cent of them did not reach the age of 50. The term *Pneumonoconiosis* was first coined by Zenker in 1860 and was meant to described disseminated pulmonary fibrosis caused by the habitual inhalation of dust. Many types of dust have been accused as causative agents. But although the irritating and sensitizing properties of organic dust are well recognized it is certain that pneumonoconiosis is never produced by organic material. It must be assumed that crystalline silicate is the most important dust in the causation of pneumonoconiosis. The clinical picture is well known. The outstanding symptom is dyspnoea which increases with the extent of the lesion. Cough is usually absent in the early stages and is rarely a prominent symptom. The susceptibility to respiratory infections is much increased in these patients. The last stages are characterized by high-grade dyspnoea and by the frequent occurrence of such complications as bronchiectasis, bronchopneumonia, pulmonary tuberculosis, emphysema, and cardiac insufficiency. The objective clinical findings are usually disproportionately insignificant in comparison with the subjective symptoms and with the roentgenological findings. In the first stage the roentgenogram shows disseminated small foci usually sharply defined. They are localized chiefly in the midfield of both lungs, and the apices are typically free. The hilum shows usually accentuation of the normal markings. In the second stage the disseminated forms are larger, the hilar shadows become more massive, and the diaphragm is frequently tented. In the third stage the individual shadows tend to coalesce, and evidence of emphysema and diaphragmatic adhesions are regularly present. Small multiple cavities may be seen which are not necessarily tuberculous. Clinical and experimental observations have proved that only silicate-containing dust produces pneumonoconiosis. Pure iron or lead dust does not. The actual danger for the worker depends on the concentration of the silicate, the amount of dust, and the length of exposure. A recent ordinance in Germany offers compensation for severe cases of pneumonoconiosis. It is necessary to arrive at a definite agreement as to which cases should be called "severe."—*Über Pneumonokoniosen in klinischer Beziehung und in ihrer Bedeutung als Berufskrankheiten, W. Brednow, Klin. Wchnschr., July 19, 1930, ix, 1363.*—(*M. P.*)

Pneumonoconiosis as Industrial Disease.—Through an ordinance of the German Department of Labor pneumonoconiosis has been included in the list of those diseases for which compensation is offered. The right to compensation exists, however, only in severe cases. It is specified that the combination of pneumonoconiosis with pulmonary tuberculosis has to be considered as the former disease as far as compensation is concerned. The term "severe form" has not been specified in the ordinance and is open to interpretation. The severity of the disease should not be judged by roentgenological appearances only, although the correct diagnosis is in most cases not possible without roentgenological examination. It is suggested that the severity should be judged less by anatomicoroentgenological characteristics than by clinical symptomatology. It is concluded that compensation should be granted whenever the clinical condition of the patient is such that his earning ability is reduced about one-third or more and if this reduction is due entirely to pneumonoconiosis or its complications in the form of tuberculosis or cardiovascular impairment. —*Bemerkungen zur Beurteilung der Silicose als Entschädigungs-pflichtige Berufskrankheit, E. Saupe, Klin. Wchnschr., July 19, 1930, ix, 1352.*—(*M. P.*)

Pneumonoconiosis.—A case is reported of a man who died with a clinical diagnosis of advanced silicosis. He had been a riveter in a boiler works for eleven years and a boiler cleaner, which included flue-cleaning, for nine years. Postmortem examination revealed pneumonoconiosis and tuberculosis. Chemical analysis of the dried material from the lungs, boiler scale and flue dust, revealed identical substances in all, including siliceous matter, ferric oxide, alumina, phosphorus, calcium and magnesium.—*Pneumonoconiosis Due to Flue Dust, W. E. Cooke, Brit. M. J., November 15, 1930, no. 3645, 816.*—(*M. E. P.*)

Pneumonoconiosis.— Morbidity and mortality records indicate that workers exposed to dust have excessive rates of respiratory diseases. Inorganic dusts produce more pulmonary disability than organic dusts. Silica (SiO_2) of the crystalline variety (quartz) is the element in dust which produces greatest pulmonary damage, and it is present in varying extent in most industrial dusts. Silicosis is insidious in its onset and progress and presents but few symptoms or physical signs in its early stages. Silicosis is almost invariably complicated by tuberculosis after a number of years of exposure, the time depending on the concentration of the dust and the percentage of free silica which it contains. Latent tuberculosis in persons entering dusty trades is usually activated after shorter exposure to siliceous dust and has more characteristics of the disease. Before a dusty trade can be absolved of its danger or stamped as dangerous, the concentration of dust and its chemical and petrographic analysis must be shown. Masks frequently give a false sense of protection. Removal of dust at its source is preferable. There has been a great reduction in the death-rate from tuberculosis among the general population during recent years, while among workers in dusty trades there has been a great increase during the same time in the death-rate from this cause.—*Dust and Pulmonary Disease, A. E. Russell, J. Am. M. Ass., December 6, 1930 xcv, 1714.* —(*G. L. L.*)

Silicosis Conference.—The International Silicosis Conference at Johannesburg considered prevention, medical aspects, prognosis, compensation, and after-care of silicosis. Silicosis becomes noticeable after widely differing periods of exposure. It is contracted through inhaling, for a sufficiently long period, dust containing percentages of silica as low or even lower than thirty. The greatest amount of harm is done by the smallest particles. The power of resistance of the individual is an important factor. The conference recommended that as soon as standard methods of assessing the dust-content of air were perfected, a complete survey of the dust concentration in dusty industries throughout the world should be made. Water applied to the point of percussion, such as axial water-feed in drills, is the only effective method in the use of water for the prevention of silicosis. Unnecessary humidifying of the air is to be avoided. Experimental evidence indicates that the solubility of silica in the tissues is the essential factor in the causation of silicosis. The progress of the silicotic condition is unfavorably influenced by infection of the lung with pathogenic organisms. Other dusts, harmless in themselves, are capable of producing serious pneumonoconiosis in a lung in which silicosis has been established. Pathologically, five stages of the disease are recognized; *First*, there is a fine bronchiolitis with an accumulation of dust filled phagocytes in the terminal bronchioles. *Second*, there is then an accumulation of dust containing phagocytes about and in the intrapulmonary lymphoid tissue and then transportation through the lymphatics into the tracheo-bronchial lymph nodes. *Third*, there is a gradual development of fibrous tissue within the mass of phagocytes and the formation of a characteristic nodule of hyaline fibrous tissue. *Fourth*, degenerative changes occur in these

foci. *Fifth*, the hyaline nodules increase in size by extension at their periphery, coalescence of adjacent nodules takes place and brings about involvement of extensive areas of the lung. Clinically, three stages can be recognized. In the first stage, respiratory symptoms may be slight or absent; working capacity may be slightly impaired; there is a departure from normal of physical signs, and the radiogram shows dense linear and nodular shadows. In the second stage, all the physical signs are increased, the nodular shadows are increased in number and size, and there is some degree of definite impairment of physical capacity. In the third stage, all signs and symptoms are grossly exaggerated, and there is total loss of working capacity. Pulmonary tuberculosis may be present in any of these stages, altering the symptoms, physical signs, and degree of incapacity. Sanatorium treatment is recommended for suitable cases. Rehabilitation schemes, such as farm settlements, have proved unsuccessful.—*International Silicosis Conference at Johannesburg, Brit. M. J., October 4, 1930, no. 3639, 567.*—(*M. E. P.*)

Silicosis.—Silicosis is a condition of pulmonary fibrosis brought about by the inhalation of fine silica dust which has been set free in working with materials containing silica. There are at least four different clinical pulmonary conditions associated with the inhalation of silica dust: (1) diffuse interstitial fibrosis of the lungs; (2) acute or subacute silicotic pneumonia, associated with the inhalation of silica dust mixed with dried alkali or soap; (3) simple chronic silicosis, as found in the stone-masons working grit-stone; and (4) silicotuberculosis, or the combination of silicosis and tuberculosis, which is the main risk in quartz mines. The "cardinal" symptom of chronic silicosis is progressively increasing dyspnoea, at first noticeable only on exertion, but in the advanced conditions obvious at all times. In many cases this is the only symptom. Of the others which may follow, pleuritis, usually of the dry variety, is perhaps the most characteristic. Chronic bronchial asthma and symptoms of emphysema occur in silicosis, but are, perhaps, more typically found as the result of inhalation of dust and fumes which do not cause fibrosis. Very rarely bronchiectasis develops, in which attacks of haemoptysis may occur. Cardiac hypertrophy and eventual myocardial failure are the rule. Generally speaking, there are no symptoms of toxaemia. Sudden death is not uncommon but some cases of chronic silicosis live to an advanced age. Diminished vital capacity is perhaps the most characteristic physical sign. Cyanosis is more marked in the acute forms and may assume a characteristic "lilac" tint. Blowing breathing is usually the earliest sign to develop and is heard at first over the upper part of the chest, but later becomes more generalized and obvious as the condition progresses. Next in importance is the dry pleural friction sounds. Râles may be present, but are not characteristic. Vocal fremitus, whispered voice and resonance are usually increased towards the apices. Finally, tubercle bacilli are absent from the sputum. A skiagram, *per se*, is not sufficient evidence of silicosis, but a typical skiagram, plus a history of exposure, with characteristic symptoms and physical signs, ensure a positive diagnosis. The fact that an abnormally large proportion of silicotic subjects die of pulmonary tuberculosis, and that this tuberculosis develops much later in life than among nonsilicotic subjects, seems abundantly proved by statistical evidence, but there is some evidence to the contrary. It is generally believed that the tuberculosis which occurs in silicotic subjects is of a low degree of infectivity, but this has not been the experience of the author. It is also generally considered that the combination of silicosis and tuberculosis leads rapidly to a fatal result, but the author has had three patients with silicosis and pulmonary tuberculosis, in whom signs of active tuberculosis disappeared. Up to the present, there is no known method of causing elimination of silica dust from the lung. In treatment, one is limited to removing the subject from his dusty environment and maintaining his nutrition. Precautions should be taken against exposure to tuberculosis. The generation of silica dust is prevented by use of water, and the removal of dust, as it is formed, by efficient forced ventilation.—*Some Aspects of Silicosis, P. Heffernan, Tubercle, August, 1930, xi, 481.*—(*M. E. P.*)

Silicosis and Tuberculosis.—The author examined 32 workers in the quartz industry, the workers being subjected to an intense concentration of dust containing 99 per cent of silicic acid. Roentgenologically 11 of the workers revealed silicosis in stage I, 3 in stage II, and 7 in stage III, while 6 were free from silicosis and 5 were questionable. The latent period of the disease averaged about one year and a definite disposition was discernible. Workers with deformities of the thorax or the spinal column showed a severe silicosis after a relatively short time. In none of the cases were tubercle bacilli found in the sputum, but one of them was a case of spontaneous pneumothorax and two postmortems disclosed silicosis without tuberculosis. The workers

were without exception strong, athletic types of men between 20 to 40 years of age. In 12 cases, in which there were found roentgenologically old and fresh tuberculous foci, there could be noted no influence of the silicosis nor of the tuberculosis upon each other.—*Silicose und Tuberkulose, W. Mascher, Beitr. z. Klin. d. Tuberk., 1930, lxxiii, 388.*—*(H. J. C.)*

Silicosis.—The South African Institute for Medical Research has made extensive studies of individuals suffering from silicosis. They observe that tuberculosis is the chief danger to be feared in dusty occupations, but the physical condition of the individual must be considered. Uncomplicated silicosis may undergo prolonged arrest for long periods, but many cases tend to progress from the earliest detectable stage to a much more advanced fibrosis. Thus, out of 219 new cases detected in 1920–1921, 156 were living at the end of the eighth year and 52 had remained stationary, but 104 had advanced to the primary or secondary stages. Seventy-five per cent of deaths which occur in silicotic miners are due directly to active tuberculosis. There exists in many, if not most, of the cases of clinically "simple silicosis" from the earliest stages, a latent element of low-grade tuberculous infection, and the presence of such latent foci may be regarded as a partial explanation of the tendency manifest in so many cases of simple silicosis for the ultimate development of active tuberculosis.—*Report of the Miners' Phthisis Bureau, Brit. M. J., January 3, 1931, no. 3652, 28.*—*(M. E. P.)*

Anthracosis, Silicosis and Tuberculosis in Coal Miners.—The word "silicosis" has come to imply a marked and formidable liability to tuberculosis, and is thus accepted in regard to compensation in "refractories" industries and in the gold-mining industry in South Africa. It is important to dissociate this implication from the word "silicosis" when it is applied to coal miners, for the latter are exceptionally resistant to pulmonary phthisis. Several quotations are given to prove this point. Accordng to a census in the United States pulmonary consumption caused 10.6 per cent of all deaths among miners and quarrymen as against 16.2 per cent for all occupied males. Figures from the *Registrar General's Decennial Supplement*, 1921, show the mean annual tuberculosis mortality of coal heavers and getters to be less than that of all occupied and retired and civilian males, metal grinders, and tin and copper miners. On the other hand there can be little doubt that silicosis without coincident exposure to coal dust is accompanied by a terrible liability to tuberculosis in middle life. In attempting to explain the exemption of coal miners it is difficult to find a satisfactory answer. It has been demonstrated that the anthracosis of coal miners is a dual condition in which the retention of coal dust in the lungs is associated with and, in all probability, due to, a state of diffuse or nodular fibrosis and lymph blockage, indistinguishable histologically and chemically from that found in the silicosis of gold miners. The degree of their silicosis is in direct proportion to the amount of exposure of the individual to silica-containing dust. There would seem, therefore, to be some sort of protection from the associated coal dust. Numerous views are presented. The explanation given here is that coal dust in the vicinity of tuberculous foci might, by absorbing the products of the growth and destruction of the bacilli, go far to prevent or diminish the inflammatory phenomena which constitute tuberculous disease. The power of finely divided coal dust to absorb and inactivate a proportion at least of the active principle on which the clinical effects of tubercle bacilli depend has been established. Whether the collection of carbon particles which have accumulated in the tissues and been bathed in the tissue juices retain their power of absorbing tubercle protein is a matter for further investigation. The probability that they can do so is emphasized by the fact that anthracotic coal miners often show tuberculous lesions of a peculiar type, characterized by an absence of the tendency to breaking down and excavation so common in adult phthisis.—*Anthracosis, Silicosis, and Tuberculosis in Coal-Miners, S. L. Cummins, Lancet, January 31, 1931, ccxx, 235.*—*(E. H. K.)*

Pulmonary Asbestosis.—Since 1924 occasional reports have been made of the discovery of certain foreign bodies in the alveoli, bronchioles, and interstitial fibrotic areas in the lungs of asbestos mill-workers. In 1929 the authors conducted autopsies on the bodies of two Negro asbestos mill-workers, discovering great numbers of "asbestos" bodies in all the pulmonary tissues, even occurring in thrombi of veins and in the peribronchial lymph nodes. There was much interstitial fibrosis. Search was then made in the sputum of asbestos workers for these bodies, by examining the concentrated sediment of sputum after digesting it in 10 per cent sodium-hydroxide solution, kept warmed to about 80°C. until the mucus was well digested and the mixture watery. These bodies have been found readily in three cases out of four, although it was anticipated that the negative case, in apparently a late state of pneumo-

noconiosis, should more readily reveal these bodies. It remains a subject for further study as to the relation of the occurrence and time of duration of these bodies in the sputum to the extent of exposure to asbestos dust, and as to the state or stage of asbestosis or other associated conditions in relation to their expulsion in the sputum. These bodies are variable in size, but for a characteristic structure, with a central filament of transparent, greenish-tinged needle-like crystal, on which is deposited, in varying quantity, nodules, blobs and segments of a homogeneous refractive substance, varying in color from light yellow to mahogany-brown. The deposit occurs more on the extremities, giving the appearance of dumb-bells or indian clubs. In the lungs these bodies have measured 12 to 100 micra long and 1 to 12 thick, in the sputum being somewhat longer and thicker. They do not take the ordinary tissue and sputum dyes. They may be stained on the slide by the prussian-blue reaction for iron, the heavier deposits taking an indigo-blue color, indicating a considerable iron-content to the deposit. It is suggested that the iron-content of the deposits on the asbestos crystals is of tissue origin, probably from the blood.—*Asbestosis Bodies in Sputum and Lung, K. M. Lynch and W. S. Smith, J. Am. M. Ass., August 30, 1930, xcv, 659.*—(*G. L. L.*)

Pulmonary Fibrosis in Asbestos Workers.—Asbestos is a "collective" term "applied to a variety of silicate minerals." The chief types of asbestos contain either (1) magnesium silicate with a little iron and little or no calcium or (2) calcium, aluminum and iron and less magnesium. Silica (combined) is present in amounts that vary from 39 to 57.2 per cent. In the industry there are many types of work, such as crushing, preparing, sieving, mixing, carding, spinning, opening, weaving, plaiting, braiding, mattress-making, etc., many of which are dusty. Seven hundred and seventy-five workers, employed from one to twenty years, have been investigated. Three hundred and sixty-three workers, selected especially to yield evidence as to the duration of exposure prior to onset, the rate of progress and the end-result of the affection, were investigated with great care with history, physical examination and X-ray (the latter in one-third of the cases). Early cases give percussion evidence of "enlarged roots;" there is moderate dulness in the more medial regions, especially in the back, and the note is everywhere thinner and of higher pitch than normal, except in the extreme apices where the note is clear and resonant. Impairment is constantly more marked on the right side, although the disease is uniformly bilateral. Expansion is limited and the signs otherwise resemble those in silicosis. The apices may descend with inspiration and rise with expiration. The most commonly encountered symptoms (and signs) are cough, cyanosis, dyspnoea, expectoration and pain. Yet advanced stages of pulmonary fibrosis (for such is the pathological end-result of "asbestosis") may be present with but slight symptoms. In early stages the films are unlike those of silicosis and present a slight "lack of translucency" called "veiling" or "a ground glass appearance." Later there is more mottling, but the lesions are not as dense as are silicotic lesions and the classification into stages is not altogether easy. Eventually there develop "gross lesions with pleural changes and displacements." The essential lesion is a chronic interstitial pulmonary fibrosis with thickened alveolar walls and the so called "asbestos bodies,"—golden-yellow bodies found in alveoli and in their walls. These bodies may reach a diameter of "100 microns, but are usually *very* much smaller." They are soluble in strong acids or alkalies. In dark field, they appear as minute spicules. They are probably made up of a central spicule of asbestos surrounded by a colloidal aggregate of proteins of the blood. They are found in the sputum and are probably pathognomonic of asbestosis. Altogether, asbestos dust must be regarded as inimical to health. Twenty-six per cent of the people examined were found to have pulmonary fibrosis which could be attributed to asbestos dust and 5.8 per cent were found to have percutory signs of fibrosis from the same cause. Signs develop usually before ten years of exposure, and the shortest exposure before development of symptoms was five years. Death occurs rarely under fifteen years of exposure. Of eight deaths recorded (not in the series studied) six were from fibrosis alone and two had had tuberculosis superadded. Attacks of pleurisy and pneumonia are not very uncommon. Bronchitis and bronchial catarrh were present in 16 per cent of the workmen examined. Pulmonary tuberculosis, including signs of old healed disease, was present in only 9.9 per cent. Very few of these were active. Although there is no evidence from this study of an increased susceptibility of asbestos workers to pulmonary tuberculosis, this question had better be left to further investigation before conclusions be drawn, chiefly because these examinations were made on people at work. Whether the several former employees now at home ill with "chest complaints" were suffering from asbestosis alone or from this disease plus tuberculosis was not ascertained. If films of asbestosis

subjects be compared with those of silicotic subjects, the amount of disease in the former will be underestimated, because of inherent differences in the underlying pathology of the two affections.—*The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers, E. R. A. Merewether, J. Industr. Hyg., May and June, 1930, xii, 198 & 239.*—(*H. S. W.*)

Primary Cancer of Lung.—The symptoms of primary bronchial carcinoma are frequently very insidious in their onset. Cough, thoracic pain, mucoid, mucopurulent, or foetid sputum, frank haemoptysis, dyspnoea, cyanosis, dysphagia, stertor and hoarseness, are due to the local lesions. Fever, chills, anorexia, nausea, vomiting, osteoarthropathy, weakness, loss of weight and cachexia, are systemic effects of the tumor. Less frequently, symptoms, such as pain, may be those of metastases, particularly in the central nervous system and bones. The physical signs are varied and are not pathognomonic of the condition. Localized bronchitis, signs of consolidation with or without râles, dulness and tenderness over a painful area, suppression of breath-sounds, diminished or absent vocal fremitus, are frequent findings. When the pleura becomes involved, there are signs of a thickened pleura or effusion. When obstruction of the bronchus occurs, the characteristic signs of atelectasis, with shifting of the heart and mediastinum toward the affected side occur. The X-ray may be characteristic, but secondary changes, such as pneumonia, bronchiectasis, tuberculosis, abscess, pleural thickening and effusion, may mask the primary lesion. Occasionally tumor cells with typical mitoses may appear in the pleural effusion. There may or may not be anaemia. In cases which have fever or necrosis, there is usually a moderate polynuclear leucocytosis. The diagnosis of early bronchial carcinoma can only be positively established by bronchoscopic examination. The cases may be divided into 10 clinical types, distinguished by their pathology, symptomatology, physical signs, X-ray, and bronchoscopic findings: *1:* An intrabronchial tumor, with cough, haemoptysis, slight dyspnoea, and slight signs of bronchial obstruction. The X-ray is negative until stenosis of the bronchus has occurred, which causes a diffuse haziness of the affected portion of the lung. The diagnosis is made by bronchoscopic examination. Metastases may occur but usually not until the lesion has extended. The recognition of this type is extremely important, as it is one of the few at the present time, which is amenable to successful treatment. It must be differentiated from early tuberculosis, mitral stenosis, and benign tumors. *2:* A peribronchial tumor, commencing in the lining membrane of the bronchus and infiltrating into the parenchyma of the lung, produces symptoms such as cough, haemoptysis, slight dyspnoea, fever, with signs similar to minimal tuberculosis. Beginning bronchial obstruction may be present. X-ray shows densities along the roots of the lung with radiations out into the parenchyma. It must not be confused with pulmonary tuberculosis, bronchiectasis, and other chronic inflammations of the lung. *3:* A solitary, rounded, sharply outlined tumor, located far out in the pulmonic field, and connected to the hilum by a thin band of infiltrated tissue, may originate from the bronchial mucosa, or alveolar epithelium. The type is rare and metastasizes slowly. It must be differentiated from benign tumors or abscesses. *4:* A scirrhous type of disease in the parenchyma of the lung occurs either as a result of the infiltration of a lobe by the tumor which becomes collapsed through bronchial obstruction, followed by a fibrosis of the parenchyma through which run strands of tumor cells, or it may be due to an infiltrating tumor which does not obstruct the bronchus, but causes a fibrous tissue reaction with resulting shrinking of the lobe involved. It is usually an epithelioma arising from the metaplasia of the bronchial epithelium or from an old tuberculous process. There is usually retraction of a portion of the chest, diminished expansion, dulness, tubular breathing or absent breath-sounds with râles sometimes present. The X-ray shows a dense shadow with a concave lower border. This tumor is of slow growth, metastasizes slowly, and is frequently mistaken for tuberculosis. *5:* This is the mediastinal type, which is more advanced but of the same nature as type 2. The X-ray shows a rounded, dense shadow extending from the hilum. Metastases are probably present in the regional lymph nodes. It is often confused with Hodgkin's disease, dermoid cyst, tuberculosis, lymphosarcoma of the mediastinal lymph nodes, and aortic aneurysm. *6:* This is a massive consolidation stage in which the tumor deeply infiltrates the entire lobe, or a large portion of it. It usually involves the pleura, shows early metastases, and may resemble acute tuberculosis, or lobar pneumonia. *7:* This phase has massive consolidation with abscess-formation. The X-ray may show cavity-formation with a fluid level within a dense mass of consolidation. The signs are those of a pulmonary abscess. Metastases are frequent. *8:* This is the bronchial-obstruction stage in which the bronchus leading to the lung or one of its lobes is obstructed by the tumor mass.

Bronchiectasis, thickened pleura, pleural effusion, necrosis of the pulmonary parenchyma, or massive atelectasis are complications of this type. In the differential diagnosis, one must consider pleural effusion, massive pulmonary fibrosis, aneurysm of the aorta, foreign body in the bronchus, gumma of the bronchus, and abscess. *9:* This is a curious and rather unusual type in which the tumor is disseminated along the course of the main bronchus in the form of patchy areas of infiltration with small, discrete nodules. Many of these discrete areas shown on X-ray may be areas of surrounding bronchopneumonia and involvement of pulmonary and pleural lymphatics. *10:* Miliary areas of carcinoma distributed throughout both lungs is a type which is very rare. The X-ray resembles closely the picture of confluent miliary tuberculosis or bronchopneumonia. The diagnosis is usually made at autopsy. The prognosis of carcinoma is uniformly bad, and the duration of the disease is from three to fourteen months after the appearance of definite symptoms. There is little that is helpful in the treatment. X-ray therapy may sometimes afford relief from pain but is sometimes attended with an aggravation of symptoms. Early bronchial tumors may be removed by aid of the bronchoscope. It is hoped that in the future, with increased skill in thoracic surgery, some advance may be made in the successful extirpation of early tumors.—*Primary Carcinoma of Lung, J. A. Miller and O. R. Jones, Tubercle, September, 1930, xi, 543.*—(*M. E. P.*)

Primary Cancer of Lung.—There are numerous and somewhat conflicting statistics regarding the incidence of primary bronchial carcinoma, but Carman states that it represents 1 per cent of all carcinoma and 2 per cent of deaths from all forms of pulmonary disease. It is more frequent in males than in females and occurs most frequently after the third decade of life. Many theories have been advanced to account for the origin of pulmonary carcinoma, but little is actually known. Most authors agree in laying emphasis upon chronic irritation of the bronchial mucous membrane as one of the underlying causes. It is possible that a neoplasm may arise from an overgrowth of a foetal bronchiectasis, or from atelectatic pulmonary tissue. It is probable that the metaplasia caused by such diseases as influenza, pneumonia or tuberculosis, is only one factor, and that there are other factors, as yet unknown, of which possibly mechanical irritation, such as dust, may be a contributing one. Primary carcinoma may be classified as follows: *1:* Carcinoma of the lining epithelium of the bronchi, which seldom produces a diffuse tumor, but usually begins at, or just below, the bifurcation of the trachea. The bronchial walls are thickened, and the parenchyma is invaded. The structure of this type of tumor is that of a squamous-cell or cylindrical-cell carcinoma. *2:* Carcinoma of the mucous glands of the bronchi, which leaves the bronchial lining relatively intact and spreads through the submucosa. Stenosis, rather than dilatation of the bronchi, is the usual result. The parenchyma may be extensively invaded by a tumor of glandular structure in the form of nodules. It is frequently complicated by sclerosis, bronchiectasis, and pleural thickening. The structure is that of an adenocarcinoma, with small cuboidal or polymorphous cells. A secretion of mucus may lead to a cystic or gelatinous appearance. *3:* Carcinoma from the pulmonary alveoli, which may be either diffuse, or multiple and nodular. The diffuse form is a massive consolidation resembling pneumonia. The nodular form may be difficult to distinguish from tuberculosis. Histologically, the diffuse type is composed of cuboidal, cylindrical, or flat cells, while the nodular form is that of papillary projections of cylindrical cells into the dilated alveoli. This latter type is rare. Carcinoma may be associated with inflammatory changes, such as exudative pneumonia, interstitial pneumonitis, and secondary pleural effusions. It may become secondarily infected and develop abscesses, or necrosis may occur. Widespread metastases occur, the more frequent sites being the pleura, other parts of the lung, liver, kidneys, lymph nodes, bone and brain.—*Primary Carcinoma of the Lung, J. A. Miller and O. R. Jones, Tubercle, August, 1930, xi, 501.*—(*M. E. P.*)

Cancer of Lung.—Statistical studies show that there has been an increase in the number of malignant intrathoracic tumors found at autopsy, and that this increase has been mainly in the pulmonary group. From 1904 to 1931, of 403 autopsies, four per cent had mediastinal growths, and one per cent pulmonary or pleural growths. From 1920 to 1929, of 670 autopsies, 5.7 per cent show mediastinal growths and 6.4 per cent pulmonary or pleural growths. The mediastinal lymph-node tumors are rapidly growing, soft and white, with a milky fluid exuding upon section. Small haemorrhages in the deeper parts, pigmented glandular remnants, ill-defined capsules, and small gray or dull yellow areas of degeneration are present. The tumor generally affects most extensively the middle mediastinal nodes, which coalesce into a large, irregular nodular collar, compressing, displacing and perforating

surrounding vital structures. Diffuse spread occurs along the line of the bronchial tree to the surface of the lung. Central spread is characterized by a massive, central growth, solid, firm, nodular and sharply defined, pressing on the surface of surrounding structures, extending outwards, but leaving the periphery of the lung free. Diffuse spread is associated with pleural effusion, bronchiectasis, purulent bronchitis, infiltrated cervical lymph nodes, and secondary metastatic deposits. In malignant disease of the bronchi, single, nodular growths are generally found, which are yellowish brown, penetrate into the bronchial wall, project into the lumen, and present a sloughing or bleeding surface. Bronchiectasis, septic bronchopneumonia and complete stenosis of the bronchi may ensue. Rarely are secondary nodules found, and invasion of the mediastinal lymph nodes is by no means constant. The nodules may be sharply localized or deeply infiltrative in character. Diffuse bronchial growths resemble miliary tuberculosis. They tend to affect one lung only and may be more marked at the base. The mediastinal lymph nodes may not be affected. In malignant disease of the lung, lower-lobe growths generally occupy the whole lobe. They are usually yellow with reddish-brown streaks due to haemorrhage. The margins of the growth are well defined due to the slow growth. The cut-surface is rather granular. Invasion of the pleura and necrosis of the centre of the tumor ultimately occurs. Upper-lobe growths may be confused with tuberculosis, as cavitation and invasion of the tracheobronchial lymph nodes frequently occurs. In whole lung growths, the interlobar fissures are completely obliterated, the bronchi are filled with plugs of growth, cavitation is not frequent, but there is pressure on pulmonary vessels. These growths often resemble testicular tumors and may even have amyloid strands suggestive of syphilitic gummata. Multiple nodules in the lung are discrete and yellow, and appear like secondary nodules in a liver. They may be confused with actinomycosis. In the sclerotic type the lung retracts against the mediastinum. Primary malignant growths of the pleura are rare. There may be a general uniform infiltration of the pleura, scattered nodules, or large pedunculated masses projecting into the pleural sac. Endothelioma implicates the whole serous membrane simultaneously and forms a thin, hard pellicle over the lung. Pleural effusion is common. Complications such as bronchiectasis, abscess, empyema, gangrene and direct extension of the growth to the heart are liable to occur. In the majority of cases, regional lymph nodes are involved; then the osseous system, liver, suprarenals, kidney, spleen, pancreas and brain.—*Malignant Disease of Lung, S. R. Gloyne, Tubercle, November, 1930, xii, 54.*—(*M. E. P.*)

Cancer of Lung.—Primary malignant neoplasms in the chest, and more especially those arising in the lungs, are extremely insidious in their onset, and may have been present for many months before their presence is suspected. Unfortunately, in a large proportion of them, dissemination into the lymph nodes of the mediastinum has resulted by the time the symptoms are sufficient to cause the patient to seek medical advice, and all hope of successful surgical removal of the growth is gone. In the lung, carcinoma appears to arise primarily in the bronchi and parenchyma. In the bronchi, carcinomatous changes may originate in the epithelial lining of the tubes, comprising the squamous-celled group which results from anaplasia or metaplasia of the normal epithelial cells, and the cylindrical-celled carcinoma in which there are very wide variations in structure; or they may originate in the mucus glands in which myxomatous degeneration is often seen. In the parenchyma, the histological appearances vary according to the type of tumor. In the diffuse pneumonic type the alveoli are occupied by cuboidal, cylindrical or flattened cells. The localized carcinomata consist of vesicles partially filled with papillary projections of cylindrical cells. Endothelioma has been described and consists of masses of cells of endothelial type. Sarcoma is generally of the spindle-shaped type, occasionally filling the hemithorax and sometimes eroding ribs and chest-wall. The onset, while generally insidious, may be initiated by a pyrexial attack commonly labeled influenza. Increasing lassitude without localizing symptoms may persist for weeks or months. Later, dyspnoea, nonproductive cough, haemoptysis, pain and dysphagia occur. In the bronchial growths, the physical signs are those of atelectasis. In the central or alveolar type, physical signs may be absent. Enlarged supraclavicular lymph nodes and paralysis of the homolateral diaphragm due to phrenic involvement may be found. Evidence of venous obstruction such as dilatation of the superficial veins of the chest, upper limbs and neck, is comparatively rare. Effusions are usually late and are often haemorrhagic. Bronchiectasis secondary to the obstruction results in some cases and its symptoms may mask those of the initial lesion. The fact that the signs are atypical of any of the common pulmonary diseases should immediately suggest the possibility of malignant disease of the lung. The different types of growths give rise to different

radiological appearances. In the growths obstructing the bronchus, the air in the occluded portion of the lung is gradually absorbed and the tissue appears opaque and wedge-shaped, with the apex of the wedge at the occluded bronchus and the base at the periphery of the lung. The mediastinum may be displaced to the affected side. In central growths definite opaque shadows, with complete absence of any pulmonary reaction around the tumor such as seen in abscesses, are found. Pleural and subpleural tumors may be differentiated by pneumothorax. The centre of the tumor may become necrotic and the picture may resemble that of a lung abscess. By means of bronchoscopy, biopsies of bronchial growths may be obtained. Thoracoscopy may be of value in differentiating pleural and chest wall growths from pulmonary tumors. Thoracic exploration, under modern conditions and preceded if possible by artificial pneumothorax, is no more serious a procedure than abdominal exploration and may result in a successful removal of a tumor. When radical removal is impossible, radium therapy should be considered. Three methods are used; application at a distance of large doses, external application of plaques, and insertion of radon seeds into the bronchi. The first method is useless; the second may cause considerable amelioration of pain; while the third procedure, although too recent a device for prolonged observation of results, suggests a distinct possibility that a curative result may be obtained, if no metastases have occurred. At least, the relief given and the prolongation of life afforded thoroughly justifies the comparatively small risk of the operation. Deep X-ray therapy has proved dissappointing, although it may deter the rapidity of development of secondary deposits in the lungs.—*Malignant Disease of the Lung, A. T. Edwards, Brit. M. J., January 24, 1931, no. 3655, 129.*—(*M. E. P.*)

Cancer of Lung.—A group of 12 cases selected from 109 in which the condition was diagnosed as primary carcinoma of the lung were studied from the point of view of metastases to the central nervous system. Three cases showed symptoms of the spinal cord due to compression following invasion and destruction of the vertebra by the metastases. Of 109 cases of primary carcinoma of the lungs investigated, 11 per cent showed involvement of the central nervous system. One of the cases came to autopsy and revealed compression and distortion of the cord without circulatory interference. The spinal cord is rarely the seat of metastasis from primary carcinoma of the lung. The onset in most of the cases showing neurological signs came on suddenly. The neurological signs may be present without any evidence of pulmonary changes. For both diagnostic and therapeutic reasons, thorough examination and roentgenograms of the chest are advisable in every case in which tumor of the brain is suspected. Metastases to the central nervous system may be single or multiple. When multiple metastases are found, the theory of tumor cells being carried by the blood-stream to the brain is favored. When a single metastasis is found, a direct extension by a backward flow of the lymph to the brain due to involvement of the cervical lymph nodes is favored. The most frequent histological types of primary carcinoma of the lung with or without metastases to the central nervous system were derived from the bronchi.—*Primary Carcinoma of the Lungs with Metastases to the Central Nervous System, C. Davison and W. A. Horwitz, Arch. Int. Med., October 1930, xlvi, 680.*—(*W. I. W.*)

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